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HISTOLOGICAL CHANGES IN BRAIN ARTERIES IN ARTERIAL HYPERTENSION AND ATHEROSCLEROSIS

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The arterial changes in hypertension and atherosclerosis cause disturbances in the cerebral blood flow (CBF). In comparison with somatic vessels the brain arteries show some peculiarities. First of all in structural and functional aspect three different compartments in the arterial part of brain circulation are to be distinguished: extra- and intracranial parts of both carotid and vertebral arteries, pial network and intracerebral circulation. In morphological point of view the cerebral arteries are comparatively less studied than somatic vessels, and investigations over autopsy materials are even less in number. So the aim of the present is to evaluate the histological changes in the different compartments of the arterial circulation of humans, suffering severe neurologic deficits, due to hypertension and atherosclerosis. The intracranial arteries of 92 autopsy cases (40 women and 52 men), including basilar, Willis circle, pial and intracerebral arteries were processed by the routine paraffin method and stained by hematoxylin-eosin, Van Gieson, PTAH, PAS and fuxeline after Weigert. The structural peculiarities of the cerebral arteries involve the relatively thin medial layer, lacking subendothelial intimal space and well-expressed internal elastic lamina. In cases of cerebral atherosclerosis in the extracerebral intracranial arteries different stages of atheroma formation were observed - subendothelial thickening, accumulation of lipid core with foam cells, cholesterol clefts and fibrous cap causing either stenosis and obliteration of the lumen or significant atrophy of the medial layer (Fig. 1a). Formation of aneurysm was found in 4 cases and thrombi - in 16. In cases of hypertension the reaction of the extracerebral vessels was represented by an autoregulatory constriction (Fig. 1b). The intracerebral arteries showed dilatation of the lumen with a thin medial layer and smooth elastic lamella when severe atherosclerosis in the extracerebral compartment existed. In hypertension the dominating lesions in the intracerebral arteries were that of plasmorrhagia, encroachment of the lumen and formation of fibrous tissue. Deposition of hyalin and perivascular hemorrhages were a common finding, too (Fig. 2a). Just in one case it was a chance to see the characteristic appearance of "goffering" (Fig. 2b). The normal function of the three compartments keeps CBF

constant in the interval 50-180 mm Hg. If the systemic blood pressure increases over the upper limit (in cases of hypertension) or if it drops below the lower limit (in cases of cerebral atherosclerosis) a reaction of complete loss of the cerebral autoregulation occurs. The changes in arterial hypertension vary from autoregulatory constriction up to decompensatory dilatation. Data coming from experimental biomicroscopy suggest that "goffered" blood vessels are the evidence, preceding the impaired autoregulation at the upper threshold. Cerebral atherosclerosis causes a decrease in the perfusion pressure distally from the stenosis and autoregulatory and decompensatory dilatation, which is expressed by torsion of the arteries and dystrophic changes in the wall. The ulcerated plaques in addition form lacunar infarctions via thrombi development arround athera particles present in the blood stream. The investigated cases appear to be the final stage of impaired CBF and the lesions of the arteries - an integrity of autoregulatory, reparative and compensatory changes.

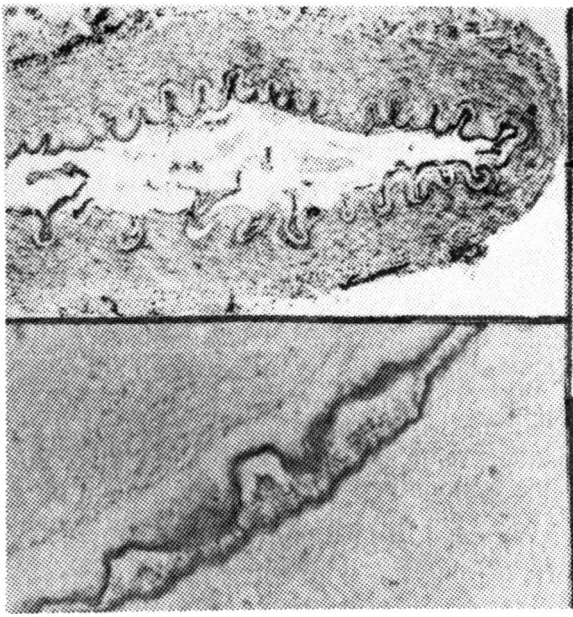

Fig. 1

Fig. 2